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THESIS FOR DEGREE OF DOCTOR OF PUBLIC HEALTH

THE CONTROL OF TYPHOID FEVER IN THE LARGER CITIES OF THE UNITED STATES.

George C. Dunham.

1921.

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When it is proposed to place any preventable disease under control, certain factors must be considered. First, is the disease in question of sufficient importance economically to justify the expenditure of the money and labor necessary to bring it wholly or partially under control? Second, is the eitiology of the disease, and the modes of transmitting the infection, sufficiently well known to permit the application of recognized scientific measures of control ? Third, which method or combination of methods, will effect the greatest reduction in the incidence of the disease ?

Any discussion of the control of typhoid fever must consider these three factors. The economic necessity of controlling typhoid fever is fully recognized at the present time. The knowledge of the etiology of the disease and the means of transmitting the infection is such that control measures can be applied in practically any community with a reasonable expectation of success.

History of Typhoid Fever.

The name of typhoid fever was first applied to the disease in 1829 by Louis who was the first to clearly differentiate typhoid fever from typhus. He based his conclusions upon the fact that in typhoid fever ulcers were found in the small intestine, and that similar lesions were never found in typhus fever.

Gerhard, an American student of Lewis, in 1837, gave a full account of the anatomical and clinical features of the two diseases. In 1842 Bartlett published a treatise in which typhoid fever and typhus were separately considered. The work of these two physicians, together with that of Enoch Hale, Shattuck, and Austin Flint, served to place the status of the disease upon a firm basis in this country.

In Great Britain, the work of the American and French observers was confirmed by the studies of Barlow in 1840, and Sir William Jenner in 1850.

During the years prior to the discovery of the typhoid bacillus a number of theories existed regarding the method of transmission of the disease.

Murchison recognized that typhoid fever could be transmitted from man to man by the excreta but unfortun-

ately included in his doctrine the theory of spontaneous generation outside the body of the infective agent. In 1862 he gave as his opinion:

> "It (typhoid fever) may be communicated by the sick to persons in health, but even then, the poison is not like that of smallpox given off from the body in virulent form, but is developed by the decomposition of the excreta after their discharge. Consequently, an outbreak...implies poisoning of the air, drinking water or other ingesta with decomposing excreta."

Much difference of opinion existed as to the comparative "contagiousness" of typhoid fever. Louis noted that typhoid fever was less contagious than typhus and other investigators alleged that typhoid fever differed from typhusin that it was less contagious than typhus. Sir William Jenner wrote in 1850:

> "If typhoid fever be contagious, it is infinitely less so than typhus. My experience leads me to regard it as contagious."

In 1856 William Budd began the publication of his studies on the methods of transmission of typhoid fever. By epidemiological studies he proved that the disease could be transmitted through the feces of a typhoid patient, by means of water or milk contamination, to healthy individuals. He believed that the

poison of the disease could be found in the diarrhoeal discharges of the patient, and that epidemics, as a rule, were caused by the contamination of the water supply with the excreta of the typhoid patients.

Budd also conceived the idea that the infective agent of the disease could be carried by persons in whom all clinical evidence of the disease had disappeared, and that these individuals were capable of transmitting the infection through their dejecta--a condition which is known to-day as the carrier state.

In 1880-81 it was shown by Eberth, Klebs, and Koch that the bacillus known to-day as the typhoid bacillus was constantly associated with typhoid fever. The typhoid bacillus was first grown in pure culture by Gaffky in 1884.

From a purely scientific viewpoint, the typhoid bacillus has never been proven to be the one and only cause of the clinical condition known as typhoid fever. However, certain facts in the life history of the bacillus and in its relation to the disease in question have led to the general acceptance of the view that typhoid fever is, in every instance, caused by the invasion of, and the multiplication within the body, of the typhoid bacillus.

The typhoid bacillus can always be found at some time during the course of typhoid fever in the blood, feces, or urine; and, except in the case of carriers, it is never found in the blood or excreta of normal persons, or in the presence of diseases other than typhoid.

Characteristic lesions are produced in the presence of the typhoid bacillus, and these lesions are accompanied by the symptom complex of typhoid fever. While the disease cannot be reproduced in the ordinary laboratory animal, it has been reproduced in chimpanzees in a form similar to that observed in man.

Immunity.

As stated above the ordinary laboratory animal is, as a rule, immune to typhoid fever as it is known in man; while, on the other hand, man is rarely completely immune to the disease in a natural state.

An attack of typhoid fever will usually confer a fairly high degree of immunity to subsequent infections. The protection thus afforded is relative and not absolute. It has been noted in virtually all epidemics that in groups of individuals, where all the members were apparently

equally exposed to the infection, only a portion of the persons thus exposed developed the disease. In some of these cases the immunity exhibited may be attributed to previous attacks of the disease, or the failure to contract the infection may have been due to mechanical factors such as the bacilli being swept through the gastro-intestinal tract without being afforded an opportunity to attack the intestinal wall. However, it is probable that some of these cases, which gave no history of a previous attack of typhoid fever, had in reality been rendered immune to the disease by a number of slight infections, no one of which was sufficient to produce the clinical symptoms. The cumulative effect of these slight infections was the production of sufficient immunity to overcome the toxins produced by a still larger number of the bacilli of similar virulence.

Scheller[1] describes a milk epidemic in which 72 people drank milk, over a considerable period of time, that was constantly being contaminated by a carrier. Of the 72 persons, 32 contracted the disease; and of the remaining 40 people, 18 were found to have typhoid bacilli in their feces or urine. Gay and Claypoole[2] report an instance where four persons drank of contaminated water and

2 developed the disease while the other 2 escaped with only slight symptoms of intoxication which disappeared within a few days.

It has often been noted that newcomers in a locality where typhoid fever is unusually prevalent often exhibit a higher incidence of the disease than do the older inhabitants who have not had the disease, indicating that a certain amount of immunity has been acquired by the older residents, probably due to a number of slight exposures.

The estimates that have been made of the number of recovered cases of typhoid fever that suffer from a second attack of the disease indicate that the protection afforded by one attack is not as effective as that conferred by such diseases as smallpox or scarlet fever. Osler[3] places the estimate of typhoid fever cases that will offer a history of the previous attack at 2.2 per cent. Sawyer and Kelly[4] found that, under conditions of massive infection, 8 and 15 per cent of their cases, respectively, gave a history of a previous attack.

The history, or lack of history, of a previous attack is, in many cases, distinctly unreliable because of the fact that many of the diagnoses were made without differentiation between typhoid and paratyphoid fevers,

and also because many mild cases of undetermined fever are wrongly diagnosed as typhoid fever.

On the other hand there is always the possibility that the second attack of the fever will be mild and pass unsuspected. The undoubted recurrences that do occur usually appear during the course of the larger epidemics where probably the infecting dose is larger and more virulent. It is probable that, after a reasonable lapse of time, any recovered case could be reinfected should a sufficient number of virulent bacilli be ingested.

The nature of the protection against typhoid fever afforded by an attack of the disease, or by vaccination, is obscure. The fact that apparently the agglutinins disappear from the blood within a few months after the development of the immunity tends to disprove the theory that the immunity is due to the action of antibodies in the blood.

It is generally assumed that the symptoms of typhoid fever are the direct result of the action of a toxin rather than the effect produced by the growth and multiplication of the bacilli alone. Further, the evidence tends to show that this toxin is one which is released by the death and disintegration of the bacilli--an

endotoxin--rather than one that is secreted by the living bacilli--an exotoxin. There has been some evidence advanced in recent years to show that while the principal toxin is an endotoxin, a comparatively small amount of exotoxin is also formed. If the present understanding of what constitutes an antitoxin is correct, it is evident that the immunity in the case of typhoid fever is not due to an antitoxin in the blood stream, provided that the term antitoxin is used in the same sense as that of diptheria antitoxin.

Certain cases of typhoid fever recover from the clinical symptoms of the disease but continue to excrete the bacilli in their feces and urine for varying lengths of time after recovery, becoming the so-called carriers of the disease. Other individuals are found to excrete the bacilli who give no history of ever having had an attack of the disease although it is probable that such cases did in reality suffer from a mild attack of the fever which passed unnoticed or was diagnosed as some other condition. Carriers afford the best example of protection against the disease and in many of these cases agglutinins cannot be demonstrated in the blood by any test now available.

The recent work of Besredka[5] has led him to con-

clude that the elements of protection against typhoid fever are localized in the cells of the intestinal wall; i.e., that an attack of the disease, or vaccination, rendered the intestinal wall impervious to the passage of the bacilli and thereby prevented them from reaching the blood stream where they could produce the general symptoms of the disease, and also protected the glands of the intestinal wall against the action of the products of the bacilli.

The gradual accumulation of evidence of this type seems to indicate that the factors that produce the immunity against typhoid fever are of cellular rather than of humoral origin.

Modes of Infection.

The primary source of the infection in any case of typhoid fever is another case, or a carrier; but the methods by which the infection can be transmitted from one to the other are extremely varied.

The ability of the infected person to infect others depends first, upon the number and virulence of the bacilli that he is excreting; second, upon the adaptability of the available means of transmitting the infection;

and third, upon the presence of non-immune persons.

The individual suffering from typhoid fever may eliminate the bacilli in his excreta during the incubation period of the disease, the active stage, and for varying periods of time after the clinical symptoms have disappeared. The number of the bacilli excreted varies from time to time in the same individual and in different individuals. In an active case of typhoid fever the dangerous period, in so far as the transmission of the infection to others is concerned, lies in the incubation period and after a clinical recovery has taken place. During the period intervening between the infection and the appearance of the symptoms of the disease, the condition is undiagnosed and the victim spreads the infection by means of the bacilli eliminated in his excreta; while, after recovery, he may become an unsuspected carrier and continue indefinitely to be a source of danger to the community.

Mild or undiagnosed cases of typhoid fever present the same elements of danger as more severe cases and in fact are often more dangerous to the community because they remain unsuspected and uncontrolled.

The methods of transmitting the infection from

the infected individual to the non-immune may be by direct or by indirect channels.

The direct method consists of the immediate transmission of the excfeta of the patient or carrier to the mouth of the non-immune person. This may be brought about by the fingers of the nurse or attendants, or by inanimate articles,such as food, that have come into direct contact with the excreta.

The indirect transmission of the infection,where the bacilli spend a part of their existence outside the human body, may be brought about by the means of water, milk, flies, food other than milk, and many other vehicles that allow the bacilli to remain viable until they reach the intestine of a susceptible person.

Persons who attend those sick of typhoid fever are more prone to contract the disease by direct contact. Gay makes the statement that it has long been recognized that nurses and physicians are more liable to contract typhoid fever than members of other walks of life. He attributes this tendency to the fact that the fingers of these individuals come in contact with the body or the excreta of the patient, and the bacilli are conveyed thereby to the mouth. Any object that comes in contact with the patient may trans-

mit the infection to others, and the acquisition of the infection by the direct method is by no means limited to the members of the nursing and medical professions.

The cases of the disease that result from direct infection are said to be more severe than those in which the infection has been acquired by an indirect method. This condition may be attributed to the larger doses as well as to an unaltered virulence of the bacilli which have not been subjected to the adverse conditions attending their existence outside the animal body.

It is probable that more infections with the typhoid bacillus are caused by the drinking of contaminated water than by any other one method. It was the first recognized route of infection; and the reduction of the typhoid fever death rate in recent years has been largely due to the improvement in the public water supplies.

While human excreta may reach a water supply in a number of ways, the two most common methods are by contamination with surface water and by percolation of sewage or contaminated water along subterranean strata or fissures. Feces and urine may be carried by the surface wash into the water supply and the surface disposal of sewage may lead to its ultimate disposal in the water supply. Instances of an accidental connection between sewer and water pipes have

occurred with disastrous results.

It is not thought that any marked multiplication of the typhoid bacilli takes place in water; but the bacilli may survive for varying periods of time, depending somewhat upon the purity of the water and the temperature. The bacilli tend to die off in water that is grossly contaminated with sewage. Warm water is more favorable for their existence than cold and they tend to disappear quickly in shallow water that is exposed to the direct rays of the sun. They will live longer in quiet water than in water that is constantly agitated. Gay states that the typhoid bacilli may live in sterile water for as long as three and one-half months; and Park and Williams estimate their life span as from two to seven days in typical river water. While most of the bacilli will disappear from contaminated water in a very short time, a few may survive for weeks and be sufficient to produce an epidemic.

The proof that water transmits the infection of typhoid fever is purely circumstantial. The typhoid bacilli have been isolated from water on only a few occasions. The evidence that water is contaminated with the typhoid bacilli rests upon the isolation of the colon bacilli as an indication of fecal contamination and upon epidemiological

studies which show that the distribution of the suspected water supply has coincided with the outbreak of the disease. The history of the disease has shown that in a great many instances the purification of the water supply has resulted in a marked reduction in the morbidity rate.

Milk, or food other than milk, as a means of the indirect transmission of the bacilli is peculiar in that the infection usually originates with a carrier and also, in many instances , these substances act as a culture media and permit the multiplication of the bacilli outside the body. Sawyer[6] quotes an epidemic that occurred at Hanover, California where a carrier prepared a dish of spaghetti which was allowed to stand over night and then browned on the following day, and served to a large number of people of whom 93 developed typhoid fever. Investigation showed that the spaghetti prepared in this manner permitted the growth of the bacilli and that the browning of the food did not require enough heat to kill the organisms. Milk, under suitable conditions as to age and temperature, offers an ideal culture media for the growth and multiplication of the typhoid bacilli.

The indirect transmission of the infection may also be accomplished by the means of the clothing of the patient or carrier. Washerwomen have acquired the infection

by handling the clothing or bed linen of typhoid patients and even that of carriers. The shoes may be contaminated with the polluted soil and the infection transferred by the hands from the shoes to the mouth.

It is highly probable that a certain amount of the infection is transmitted by flies from infected feces to the food. Studies of the epidemics that occurred during the Spanish-American War by Reed, Vaughan and Shakespeare[7] tended to show that the transmission of the typhoid bacilli by this method was probable, although no proof to support the hypothesis was offered. Later the work of Hamilton[8] showed conclusively that the house fly, *musca domestica*, did contain the typhoid bacillus in some instances. ~~His~~ Her work was confirmed by Klein and Fricker[9]. The flies may carry the bacilli on the exterior of their bodies, or in the gastro-intestinal canal from which they are excreted in the feces. It is doubtful if the bacilli multiply within the body of the fly; but Whipple states that, under artificial conditions, the fly may retain the living bacilli in its body for as long as twenty-three days.

Discussion of the Methods of Transmitting the Infection.

A marked difference exists in the number of cases of typhoid fever due to the various methods of transmitting the infection in different epidemics. Under the conditions that surround the epidemiological study of an epidemic there is usually a fairly large residue of cases in which the source of the infection remains undetermined.

Osborn[10], in a study of the sources of the infection in 2007 cases of typhoid fever occurring in Massachusetts during the years of 1918-19 found that in only 26.6 per cent. of the cases could the source of the infection be shown. The data obtained in this survey is presented in Table No. 1.

Table No.1

Sources of Infection of Typhoid Fever Cases Occurring in Massachusetts during the years of 1918-1919. 10.

Classification	Number of cases 1918	Number of cases 1919	Total cases	Percentage of cases investigated.
Direct contact with a typhoid case.	98	123	221	13.3
By milk infected by a carrier	46	56	102	6.1
By typhoid carrier other than milk	4	10	14	0.8
Milk infected by a clinical case.	6	0	6	0.4
Food outbreak, probably a carrier.	0	10	10	0.6
By water containing large numbers of colon bacilli.	10	80	90	5.4
Totals	164	279	443	26.6
Unknown source pf infection.	588	634	1222	73.4
Totals.	752	913	1665	100.0

O'Donnell,[11] in a more extensive study of the source of the infection of typhoid fever in Massachusetts--from 1913 to 1919--concluded that flies, food (exclusive of milk), privies, and sewage were relatively unimportant in the spread of typhoid in that State. He found that few cases were due to water, especially in recent years; and in general, no municipal water supply in the State could be considered as a dangerous source of typhoid fever.

O'Donnell further concluded that milk as a means of spreading the infection of typhoid fever was responsible for 8.1 per cent. of the total cases that had been reported in Massachusetts during the preceding ten years, 1910 to 1919 inclusive.

Howard,[12] while making a study of the typhoid fever situation in Baltimore, observed that a much larger percentage of the cases in the years 1918-1919 were reported as being due to contact than in the years 1916-1917. He attributes this to the fact that more complete histories of the cases were obtained in 1918-1919 than in the two preceding years. He also reported that in 1916-1917 the infection was transmitted by the milk supply in 4.52 and 7.9 per cent. respectively of the reported cases; and that in the years 1918-1919, no cases were found to have been due to this source. This change was probably due to the enforcement of

a new ordinance controlling the milk supply in the last two years. The data obtained by Howard in his study of the sources of infection in typhoid fever in Baltimore are shown in Table No.2.

Table No. 2

Sources of Infection of Typhoid Fever in Baltimore during the years of 1916 to 1919. (12)

	1916		1917		1918		1919	
Sources of Infection.	Cases	Percent of cases	Cases	Percent of cases	Cases	Percent of cases	Cases	Percent of cases
Infected out of City.	125	16.42	142	26.10	53	17.55	50	17.06
Contact with recent cases.			21	3.86	39	12.58	36	12.28
Contact with remote cases.			15	2.75	22	7.90	23	7.84
Neighborhood Infection.							5	1.70
Spring, wells, and streams.	4	0.51					12	4.09
Carriers.	14	1.80			5	1.65		
Milk.	34	4.52	43	7.90				
Laboratory					1	0.33	2	0.67
Self infection.			4	0.73	4	1.32		
Probable outside.					16	5.22	24	8.19
Probable inside.					20	6.26	14	4.74
Unexplained.	597	77.13	319	58.83	142	47.02	127	43.24
Totals.	774	100.00	544	100.00	302	100.00	293	100.00

Freeman[13] made a survey of the sources of typhoid infection in twenty-four cities in the Ohio river valley in 1914-1916. This survey included the collection of information regarding the prevalence of typhoid fever during the five years prior to this time in all the cities of more than 25,000 population in the Ohio River valley, except Pittsburgh, Cincinnati and Louisville.

The cities were rated in accordance with the purity of their public water supply and the proportion of the population that were users of the public supply.

Freeman found that in general the typhoid fever death rates varied regularly in inverse proportion to the water supply rating. A summary of the results obtained is given in Table No.3.

Table No.3

Summary of Water Supply Ratings by Groups and the Typhoid Fever Death Rates for the same Groups.[13]

Group rating	Number of cities	Total population of group in 1915	Death rate per 100,000 for each group. 1910 to 1914
90-100	8	847,360	21.1
80-90	8	459,130	36.5
70-80	3	119,796	22.3
50-70	2	171,074	37.7
0-50	3	102,456	47.3

From the observations made in this survey Freeman concluded that the major factors in the typhoid causation in these cities were polluted water and the improper disposal of human excreta, and that the principal minor factor was the lack of pasteurization, or the improper pasteurization of the milk supply.

In an investigation of a typhoid epidemic in Omaha in 1909-1910 by Lumsden[14], it was found that the public water supply had become polluted to a dangerous extent by sewage; and that of 103 cases of typhoid fever studied 95 per cent. had drank of this water at some time during the thirty days prior to the onset of the disease. As no other common sources of the disease were found, it was concluded that the infection in this epidemic was, to a large extent, water-borne.

A study of the typhoid situation in North Yakima, Washington was made by Lumsden[15] during the years 1909 to 1911, inclusive. Lumsden concluded that the high rate of the prevalence of typhoid fever in North Yakima in the summer and fall of the years 1909 and 1910 was due, for the most part, to the local dissemination of human excreta from insanitary privies, privy vaults, cesspools, septic tanks, and bedsides of the sick to the mouths of susceptible persons by fingers, flies, foods, and water; and that the

amount of infection introduced into the city by the public water supply, milk supply, fruit and vegetables was relatively unimportant. On the other hand, during the year of 1911, it was found that in the majority of the cases the infection could be traced to the public water supply which had become contaminated by the addition of water from a local pond.

Lumsden and Anderson[16] in their study of the origin and prevalence of typhoid fever in the District of Columbia found that in 1906, and for some years prior thereto, the water supply from the Potomac river was a factor--and at times, a very considerable factor--in the spread of the disease. Filtration of the public water supply was begun in 1905 and after the autumn of 1906, it played a relatively unimportant part in the causation of typhoid fever in Washington.

Investigations along other lines showed that during 1906, 1907 and 1908, outbreaks of typhoid fever occurred which could be definitely traced to milk-borne infections in about ten per cent. of the cases. It was also found that raw shell fish contributed, to a large extent, to the prevalence of the disease during the winter months; and that during the summer months, flies, raw vegetables and fruits played a definite though minor role in the spread of the disease.

As a result of their five years of study, Lumsden and Anderson concluded that the greater part of the typhoid fever in Washington was derived from the dejecta of typhoid fever patients and typhoid carriers within the city and spread by means of milk, green vegetables and other foods, and by fingers and flies.

During the years that have elapsed since it was discovered that typhoid fever was an infectious disease and that it was transmitted in the excreta of the human body, innumerable epidemiological studies have been made to determine the routes by which the infection traveled from the sick to the well. It would be impossible to cite even a small portion of them here. The few that have been mentioned have been quoted to show the wide variation in the modes of transmission of the infection. It would be futile to attempt to ascribe any definite percentage of the cases to any one source of infection.The source of the infection in every epidemic depends upon the local sanitary conditions and any epidemiological study must take into consideration all the factors that bear upon the possible means of transmitting the infection in that particular community.

It is noteworthy, however, that in recent years the steady year-to-year incidence of urban typhoid fever is

usually due to other causes than that of water-borne infections. This condition may be attributed to the fact that the public is gradually being taught that the disease can, to a large extent, be controlled by the purification of the water supply and the proper disposal of the sewage. Also the water supply and the sewage disposal system of a community are usually under the direct supervision of public officials while the sanitation of local premises and the handling of food supplies are more largely a personal matter.

Varying estimates have been made as to the amount of typhoid fever that is due to causes other than contaminated water. Freeman[13] states that the installation of universal sewerage in the presence of a pure water supply will reduce the typhoid fever death rate to about 10 per 100,000 population in the northern cities and to about 20 per 100,000 in the southern cities. Johnson[17] estimates that pure water supplies and efficient sewage disposal will reduce the typhoid death rate to about 14 per 100,000 population.

Explosive outbreaks of typhoid fever have occurred from time to time; and in the majority of such epidemics, the source of the infection was traced to polluted water

supplies, due to the lack of purification, or the inefficient purification of the water supply.

Horton[18] investigated an epidemic at Herkimer, New York, and found that 155 cases of typhoid fever occurred between September first and December first, due to the use of unpurified water from an canal.

Another instance of this type is an epidemic which occurred at Tonowanda, New York, in the summer of 1919, when 256 cases of typhoid fever were reported, following the breaking of the intake pipe of the water system and the admission of polluted water to the mains. The epidemic was immediately checked by chlorination of the water supply.[19]

In an epidemic in which the primary infection is water-borne, it does not necessarily follow that the water must have been drunk by every person who eventually contracted typhoid fever during the course of the epidemic. The water that is used to wash vegetables, fruits, or utensils used in caring for the milk supply may transmit the infection. Also, in communities where the standards of sanitation are low, an epidemic that was originally due to the temporary contamination of the water supply may become an epidemic due to the transmission of the bacilli by food, or by direct or indirect contact with the primary or secondary cases of the disease.

In the majority of the cities of the United States,

the purification of the water supply and the proper disposal of the sewage are effective in preventing the transmission of the typhoid bacilli by these means. Nevertheless, in practically all cities, there occurs a fairly constant annual typhoid fever death rate. This is the so-called residual or endemic typhoid fever and its occurrence must be attributed to sources other than the water supply. Of recent years, it has been shown that the endemic typhoid fever is largely a question of contact, direct or indirect, with patients or carriers.

Carriers.

Epidemiological studies have shown that in many communities the carrier of the typhoid bacillus is responsible for the production of a small though fairly constant number of cases of typhoid fever each year.

From the standpoint of the transmission of the disease, the carriers may be divided into two general classes: i.e., those that eliminate the bacilli in the feces and those that eliminate them in the urine.

In the former class it has been fairly well established that the foci of infection are located in the intestinal tract or its appendages, usually in the gallbladder;

while in the latter, the source of the infection is situated somewhere along the urinary tract, usually the kidney.

Other foci of minor importance may exist in various parts of the body, as in the case of periostitis or otitis media due to infection with the typhoid bacillus.

The majority of the carriers present a history of having had typhoid fever at some time; but, nevertheless, a considerable number are found who have no knowledge of ever having suffered from the disease although, in these cases, there is always the possibility that a mild or abortive case of typhoid fever passed undiagnosed as such; or that a case of typhoid fever was diagnosed as some other disease.

The relation of the carrier to an actual attack of the disease permits of still further classification. Sacquepee[20] has classified typhoid carriers as follows :

Group I. Precocious or incubation carriers. These consist of those individuals who have ingested the typhoid bacilli and are beginning to eliminate them without having as yet shown any symptoms of typhoid fever.

Group II. Carriers that have recovered from typhoid fever, who may be subdivided in respect to the duration of carrier state.

A. Convalescent carriers. Individuals who eliminate the bacilli up to three months after recovery.

B. Chronic carriers. Individuals who eliminate the bacilli from three months after recovery to indefinite periods of time.

Group III. Healthy or paradoxical carriers, with no history of typhoid fever.

It is very difficult to estimate the number of typhoid carriers that are present in the general population of the country. Gay[21] estimated that five per cent. of the recovered cases of typhoid fever become chronic carriers. Osborn[10] found that in Massachusetts in 1919 the recovered cases yielded 1.4 per cent. of carriers; and O'Donnell, working in the same State, estimated that about 2 per cent. of the recovered cases become carriers.

The laborious methods required for the detection of carriers by means of laboratory tests practically prohibit a survey of the entire community for carriers. Some of the factors that tend to prevent the detection of the typhoid carrier are the intermittent excretion of the bacilli, the difficulty of isolating the bacilli from the stools and, in a certain number of cases, the absence of any history of an attack of typhoid fever. Epidemiological studies will reveal

only those responsible for active cases; and consequently, these are usually the only ones apprehended, with the exception that, in a few instances, food handlers are examined bacteriologically and carriers detected by this method.

It has been shown that a certain number of persons will excrete the bacilli during the incubation period of the disease, and constitute a particularly dangerous source of infection as the fact that they are carriers is never suspected.

Many of the recovered cases continue to excrete the bacilli for a varying length of time after recovery but eventually the elimination of the bacilli ceases. These constitute the so-called temporary carriers. Recovered cases that cease to excrete the bacilli within three months after recovery are usually referred to as temporary carriers; while those that continue to eliminate the bacilli after this period of time has elapsed are designated as chronic carriers.

From time to time surveys have been made to determine, in so far as the methods at hand would permit, the percentage of typhoid carriers in the general population of the different communities. The results of three such surveys[22] are given in Table 4.

Table No.4

Occurrence of Typhoid Carriers in the General Population.

Author	Date.	Locality	Persons examined	Percentage of carriers
Rosenau Lumsden and Kastle	1909	Washington	993	0.30
Prigge	1909	Saarbrucken	10,841	0.29
Muller, P.T.	1916	Germany	20,019	0.80

NOTE. These include typhoid, paratyphoid and dysentery carriers.

Gay states that an estimate of three carriers per 1000 in the general population must be regarded as extremely conservative when the intermittence of the carrier state and the difficulties in detecting the bacilli in the excreta are considered.

During an intensive antityphoid campaign conducted in the Trier district in Germany from about 1904 to 1918, seventy-one carriers were detected.[23]

During the world War, efforts were made in many of the organizations of the United States army to examine food handlers for the presence of typhoid carriers. Ninety-eight carriers were reported, but the number of the men examined is

not known.[24]

The relation of the carrier state to the history of previous typhoid is important because of its bearing upon the results of an epidemiological study of the epidemic. Osborn and Beckler[25] found that of the 13 carriers discovered in Massachusetts in 1919, 3 gave a negative history of typhoid fever.

The history of previous typhoid fever given by 71 carriers during the anti-typhoid campaign in the Trier district[23] in Germany is outlined in Table No.5.

Table No.5.

History of Previous Typhoid in 71 Carriers.

History	Number of cases.
No history of typhoid	3
Doubtful history of typhoid	16
Typhoid less than six months before	12
Typhoid between six and twelve months before	9
Typhoid between one and two years before	6
Typhoid between two and five years before	6
Typhoid between five and ten years before	7
Typhoid more than ten years before	12
Total	71

If the carrier state can exist in the presence of the immunity conferred by an attack of the disease, it can be assumed that should a person who has been artificially immunized against typhoid fever by vaccination ingest the typhoid bacilli he may become a carrier and thus increase the number of the carriers who do not present a history of an attack of the disease.

The importance of this type of carrier is obvious when the difficulty of examining every person in the community is compared with that of examining only those that present a history of an attack of typhoid fever.

The importance of the typhoid carrier as a source of infection is being emphasized more and more in the studies of endemic typhoid fever. The amount of harm that a carrier may cause depends upon his relation to the lives of others. As a rule, he transmits the infection by contamination of food supplies, and he is responsible for the occurrence of individual cases or epidemics in accordance with the intimacy of his contact with others and his occupation.

The percentage of the total number of the cases in any epidemic that are due to typhoid carriers will vary according to the nature of the local sanitation and the customs of the people. This percentage has been estimated at from 20 to 30 per cent. in Europe; while in this country,

Osborn and Beckler[25] estimate that at present more than one-half of the cases in Massachusetts are due to typhoid carriers, and Osborn[10] is of the opinion that the purely endemic typhoid fever in Massachusetts is wholly due to carriers.

According to the laboratory records of the 71 carriers in the district of Trier, Germany, the following relationships with regard to secondary cases were exhibited; 30 of the 71 gave no secondary cases before the carrier state was determined; 42 of the 71 gave no secondary cases after the carrier state was determined; 21 of the 71 gave no secondary cases either before or after the carrier state was determined; 41 of the 71, before the carrier state was determined, gave 76 direct secondary cases and 58 indirect;-- a total of 134 cases, or 3.2 cases per carrier; 29 of the 71, after the carrier state was determined, gave 55 direct secondary cases and 41 indirect; a total of 96 cases, or 3.3 cases per carrier.

Thus the 71 carriers have been interpreted as causing 230 cases of typhoid fever. If the rates are compared for the secondary cases per carrier, before and after detection, it would appear that the determination of the carrier state had not changed the rate. It is impossible to say how many new cases each carrier was responsible for before the carrier state was determined, but it is prob-

able that it was much greater than was absolutely proven. But after the carrier state was determined and his location made a matter of record the number of cases attributed to him while not complete is much more nearly accurate.

That in certain instances explosive outbreaks of typhoid fever have been due to carriers is well known. An example of such an outbreak is one reported by Leake[26] as having occurred at Hopewell, Virginia in July 1920. Here ninety persons partook of a dinner for which a portion of the food had been prepared by a cook who, at the time, was in the incubation stage of typhoid fever. Subsequently, from 5 to 23 days after the dinner 49 of the 90 persons became ill enough with the clinical symptoms of typhoid fever to be confined to bed, and one fatality resulted. There were nine other cases of illness of from 3 to 17 days' duration which were not severe enough to warrant confinement to bed. If all the cases are assumed to be typhoid, the incidenceof the infection would be 64 per cent. This series of cases afford an example of an epidemic traceable to a carrier of the incubation or precocious type.

The percentage of the cases that can be attributed to carriers has increased in recent years. This increase is due in part to increased efficiency in the methods of detecting the carrier, but more to the fact that the reduction

of the incidence of typhoid fever has been largely at the expense of the water-borne infection. Garbat[27] has estimated that 55 per cent. of the typhoid fever of to-day can be traced to carriers, directly or indirectly.

The Prevention or Control of Typhoid Fever.

The control or the prevention of typhoid fever involves the control or the elimination of the means of transmitting the infection from the patient or carrier to the non-immune person.

The primary source of the infection in typhoid fever is always another case of the disease, or a carrier; but the methods of prevention employed at the present time admit of the classification of the means of transmitting the infection to a susceptible person into two general classes: the water-borne infection and the infection transmitted by contact. The latter class can be subdivided into direct and indirect methods of transmission.

Classification:

1. Water-borne.
2. Contact with carrier or patient.
    (a) Direct contact by means of fingers, etc.
    (b) Indirect contact by means of milk, food other than milk, flies, clothing, etc.

The results of a large number of studies of the typhoid situation have indicated that the first step to be taken in the control of the disease is usually that of water purification and proper sewage disposal. An analysis of the literature on the various methods of transmitting the infection affords evidence that the major portion of the incidence of typhoid fever in the past has been due to water-borne infections; but that many communities have reached a point to-day where the proper attention to the purification of the water supply and the effective disposal of the sewage has eliminated this source, and that the residual or endemic typhoid fever that remains is mainly due to contact, direct or indirect, with patients or carriers.

Purification of the water supply offers the simplest and most effective method of reducing the incidence of typhoid fever to a certain point, generally considered to be between 10 and 20 deaths per 100,000 population per year. The reduction of the typhoid rate below 10 per 100,000 is accompanied by many difficulties and the progress is slow. The methods of effecting a further reduction consist mainly of the prevention of the spread of infection by direct or indirect contact with active cases or with carriers. This involves the detection and control of each case of typhoid, mild or severe, and of each carrier. It necessitates the supervision

of the production and distribution of milk and other food supplies, and the sanitation of the privies, and the prevention of access by flies to infectious materials.

The reduction of the incidence of the water-borne typhoid fever in a city by means of the purification of the water supply depends largely upon the number of the inhabitants that use the purified supply. However, once the pure water supply is installed, the use of the wells and private supplies within the radius of the distribution of the public water supply will gradually be abandoned. Consequently, in most cities the typhoid fever due to water-borne infection can be practically eliminated.

The effect produced upon the public mind by the marked reduction of typhoid fever by the proper handling of the water supply will generally aid the health authorities in the enforcement of other measures which have for their purpose the reduction of the incidence of typhoid fever due to other causes. In addition to the still further extension of the water supply and sewerage, such measures usually include the control by the health department of the milk supply, the location and control of carriers, and the elimination of insanitary privies, together with such other measures as may be necessitated by the local conditions. The gradual reduction in the number of deaths due to endemic

typhoid fever by these measures of control is shown in the charts of the typhoid fever death rates of Philadelphia, Columbus, Cincinnati and Baltimore. (Charts Nos. 1, 2, 3, and 4).

The general effect of the purification of the water supply upon the typhoid fever death rate in these cities was the same; i.e., that of effecting a marked reduction; but the decrease varied to a considerable degree in the different cities. Of the four cities cited, Cincinnati, Philadelphia and Columbus show an abrupt decrease in typhoid fever death rates; while in Baltimore, the decrease was not so pronounced. The results obtained in the three former cities indicate that the infection was water-borne for the larger part; while in Baltimore, it was evident that factors other than water played an important role in the transmission of the disease. The more gradual decrease in the typhoid death rate that took place in these cities in the years that followed the purification of the water offer typical examples of the difficultiesthat are involved in the eradication of the sources of infection that produce the purely endemic typhoid fever.

The enforcement of proper laws for the pasteurization of all the milk sold in the city would place an effective block

between this source of infection and the non-immune consumer. This is well illustrated in the case of Baltimore where 4.52 per cent. of the cases of typhoid fever reported in 1916 , and 7.9 per cent. of those reported in 1917 were directly traceable to the milk supply and following the putting into force of a suitable milk ordinance in 1917, no cases of typhoid fever due to this source occurred in 1918 or 1919.

The presence of insanitary privies in a city is also an important factor in the transmission of the infection and the elimination of such privies has been effective, in some instances, in producing a marked decrease in the typhoid fever death rate. At Jacksonville, Florida, a large number of insanitary privies were to be found prior to 1911. During that year, a large proportion of these privies were eliminated or rendered fly-proof. This measure was followed by a marked reduction in the death rate from typhoid fever, the rate falling from 102.3 per 100,000 in 1910 to 31.1 in 1912.

The official supervision of the production and distribution of raw vegetables, shell fish and similar foods would tend to prevent the occurrence of the disease, due to transmission of the infection by these means.

In the last analysis, the control of endemic typhoid fever is the control of the typhoid carrier and the typhoid patient. An effective control of the carrier or the patient involves, first of all, their detection, and then adequate supervision.

The detection of the typhoid patient, which is comparatively simple, means the prompt diagnosis and report of all cases of typhoid fever, both the mild or abortive type and the more severe cases. Once reported, these cases can, in most cities, be adequately controlled by the existing laws.

The first step in the detection of typhoid carriers should be the requirement that the feces and urine of all convalescent typhoid patients be examined for the presence of the typhoid bacilli at frequent intervals during convalescence and after recovery until it can be determined that the individual is or is not a carrier.

The measures that would be necessary for the detection of the chronic carrier in the general population render their isolation extremely difficult and, to a large extent, impractical.Even the detection of such chronic carriers as might be connected with the food supply of the inhabitants of a city would involve not only the recognized

measures of examining those whose work necessitated the handling of the milk supply and the food served in restaurants and hotels, but also the examination of all housewives, servants, nurses, workers in shops and stores that sold food supplies, makers and purveyors of confections and many others. If all the carriers connected in any manner with the food supply could be detected, there would still remain a certain number in other occupations who could transmit the infection by other means,such as clothing.

Once the carrier is located, the question of his supervision and control arises. When only a small number have been located, their employment in positions where harm will result because of such employment can, in general, be prevented. Up to and including 1910, Baltimore had 24 carriers under control of the Health Department[12] and the State of Massachusetts had located 51.[10]

It is almost impossible, under the present conditions, to make even an approximate estimate of the number of carriers that could be expected to be present in the population of any city. It is probable that 5 per 1000 of the general population would be a conservative estimate. Assuming that this figure is correct, the population of Baltimore would contain 2500 carriers. The attempt to control this

number of carriers by the supervision of their employment and the restriction of such employment to positions where they could not transmit the disease to others would assume the aspects of an important economic problem.

The law of New York is a typical example of the legal restrictions that can be placed upon the proven typhoid carrier at the present time.

This law defines a typhoid carrier and directs that upon the detection of such a carrier, the local health officer shall report the case to the State Health Department, giving the full name, age, address and occupation of the carrier and shall inform him or his guardian that he is a carrier and issue certain instructions as to the precautions that he shall take.

Section 3 provides that:

> When an outside toilet is used regularly by the typhoid carrier, it shall be equipped with a watertight container so screened as to exclude flies; and the removal of the contents for disposal should be in accordance with the instructions issued by the health officer.

Section 4 provides that:

> No typhoid carrier may engage in any occupation involving the handling of milk or other food product to be consumed by others. Should a typhoid carrier be discovered upon a dairy farm, the health officer may prohibit the sale of the milk, cream or butter except under the conditions stated in the Sanitary Code.

Section 5 provides that:

> No typhoid carrier shall permenently leave the community in which he resides without notification to the health officer who is to be informed of his destination, including his new address. The health officer should immediately notify the State Health Department of the change of address.

Section 6 provides that:

> The local health officer shall visit each typhoid carrier within his jurisdiction at least once monthly in order to determine whether his instructions are being complied with; and once each quarter shall render a report regarding such carrier to the State Department of Health, upon a form prescribed for that purpose.

Section 7 provides that:

> The health officer shall cause samples of the discharges from each carrier to be examined bacteriologically at intervals at a laboratory approved by the State Department of Health, and a carrier may be regarded as recovered and be discharged from observation when four successive samples taken not less than seven days apart, shall have been found not to contain typhoid bacilli;- except that no negative report shall be considered if the specimen has been delayed in transit; and in no instance, if more than two days have elapsed between the collection of the specimen and its examination.

It will be noted that the above law prohibits specifically and provides penalties for only the employment of a carrier in the handling of milk or milk products. This leaves open many channels whereby the carrier may transmit

the infection to others.

It is probable that even with the mild restrictions imposed upon the carrier by this law would be very difficult to enforce in any considerable number of cases. No law can be widely enforced unless it is sustained by public opinion and the education of the public up to the present time has not been such as to lead to the formation of public opinion in favor of restricting an individual whose only crime is that he is a carrier of the typhoid bacillus.

In the majority of cases, the spread of the infection can be controlled much more effectively by securing the coöperation of the carrier and his relatives than by legal measures alone.

Various methods of treatment have been advocated from time to time in attempts to cure the carrier state. No method has as yet been devised that will cure every case. The removal of the gall bladder seems to be the most effective in the treatment of the intestinal carrier; while in the urinary carriers, the removal of the infected kidney has been successful in some cases.[28]

It is evident that the present methods of control of carriers and the consequent contact infections will

not permit the complete eradications of typhoid fever. An irreducible amount of endemic typhoid fever will remain as long as the carriers of the disease and non-immune persons are allowed to come into direct or indirect contact.

In addition to the control of the factors that permit the typhoid bacilli to reach the non-immune individual, there still remains, as an element of protection, the method of rendering the individual immune to the infection, i.e., vaccination with typhoid vaccine.

Vaccination against Typhoid Fever.

The basis upon which the practice of antityphoid vaccination has been built is the observation that an attack of typhoid fever confers an immunity which affords protection to a greater or less degree against subsequent attacks of the disease.

The harmlessness of inculating the killed typhoid bacilli into a human being was demonstrated in the year 1896 by Wright in England, and Pfeiffer and Kolle,[29] in Germany. In 1897, Wright suggested the use of the vaccine as a preventive measure in the British Army and in hospitals. This marks the beginning of the use of vaccine to prevent typhoid fever.

Soon after came the Boer war in 1900, and the use of the vaccine among the British troops destined for South Africa was authorized by the British War Office. From the year 1900 to 1903 there were 19,096 British soldiers vaccinated, according to available statistics.

When the morbidity and mortality rates for the vaccinated and unvaccinated British soldiers in the South African campaign and for the American soldiers in the Spanish-American war are compared, it will be seen that the rates are decidedly lower among the vaccinated men than among the unvaccinated. The results observed are tabulated in Table No.6.[30]

Table No.6

Comparison of the Morbidity and Mortality Rates of Typhoid Fever among Vaccinated and Unvaccinated Soldiers.

	Total strength	Cases	Morbidity rate per 1000	Deaths	Mortality rate per 1000
British Army Boer War 1900-1903	380,605	57,684	151.56	8022	21.08
American Army Spanish-American War	107,973	20,738	192.60	1580	14.62
Vaccinated men Boer War	19,096	226	11.84	39	2.04

In spite of the low rates among the vaccinated soldiers many adverse reports were received by the British War Office regarding the use of vaccine for the prevention of typhoid fever among the troops; and for a time following the Boer war this measure was discredited in many quarters. Shortly thereafter the British War Office appointed a commission to reinvestigate the whole subject of antityphoid vaccination. The favorable report of this commission did much to encourage the use of the vaccine in the years that followed.

Voluntary vaccination was authorized in the American Army in 1909. The experience in the mobilization camps in 1898 had served to show that the sanitary measures that were practical during the war would not, unassisted, prevent the occurrence of epidemics of typhoid fever.

The report of Reed, Vaughan and Shakespeare[7] showed that typhoid fever appeared in over 90 per cent. of the volunteer regiments within eight weeks of going into camp; and in some regiments of regular troops it appeared in from three to five weeks. It also appeared in both large and small encampments in the Northern as well as in the Southern States. They stated their belief that

> "..with typhoid fever as widely disseminated as it is in this country, the chances are that if a regiment of 1300 men should be assembled in any section and kept in a camp, the sanitation of which was perfect, one or more cases of typhoid would develop."

When the troops were mobilized on the Mexican border in 1911, orders were issued for the compulsory vaccination of all the personnel against typhoid fever. The results obtained by this measure in the reduction in the typhoid fever morbidity and mortality rates at the camp at San Antonio, as compared with those obtained in a camp of similar size at Jacksonville during the Spanish-American war, are shown in Table No. 7.[30]

Table No.7

Typhoid Fever in Two Military Camps (1899 and 1911).

	Total Strength	Cases with positive diagnosis of Typhoid fever	Deaths from typhoid fever	Deaths from other causes
1898.Camp at Jacksonville	10,759	1729	248	33
1911.Camp at San Antonio	12,801	2	0	11

The small number of deaths due to other causes than typhoid fever indicate that the sanitary conditions were similar in both camps, in so far as crowding an opportunities for the transmission of infection by contact was concerned. The length of time in camp was approximately the same in each case. While the soldiers were encamped at San Antonio, there occurred 49 cases of typhoid fever with 19 deaths in the civilian population of the city. The soldiers mingled freely with the civilian

population, eating at the public eating places and drinking the same water as the civilians.

During the World War, from April 7, 1917, to November 11, 1918, approximately 4,000,000 men served in the United States Army. On November 1, 1918, there were 1,971,000 men in Europe and 1,663,000 in the United States, or a total of 3,624,000 men under arms on that day alone. During this period of time there were 1083 cases of typhoid fever among the troops, with 158 deaths.

The morbidity rate for typhoid fever during the World War was 0.34 per 1000; during the Civil war it was 70.19; and for the Spanish-American war, it was 91.22. Had the rate for the Civil war prevailed during the World War there would have been 226,001 cases; and if the Spanish-American war rate had prevailed, there would have been 291,637 cases. Actually, there were 1083 cases.

While undoubtedly the sanitary conditions were better, particularly in the camps and back of the lines, during the World War than during the Civil war and Spanish-American war, nevertheless, in any movement of large bodies of troops, or in the concentration of troops in large camps, contact of a nature that will permit typhoid carriers to transmit the infection is unavoidable. Therefore, the re-

duction of the incidence of typhoid fever among the soldiers during the World War must, to some extent at least, be attributed to the artificial immunity conferred by antityphoid inoculation.

Table No.8.

Typhoid Fever Death Rates per 1000 Population for Similar Age Groups in Military and Civil Life.

Year	Army death rate per 1000	Age 20-29 years ++	
		Civilian Death rate per 1000	Male Civilian death rate per 1000
1900	0.43	0.46	
1901	0.64	0.42	0.54
1902	0.86	0.40	
1903	0.28	0.35	
1904	0.27	0.35	
1905	0.30	0.32	
1906	0.28	0.32	
1907	0.19	0.28	
1908	0.23	0.28	
1909±	0.28	0.23	
1910	0.16	0.27	0.34
1911±	0.09	0.23	
1912	0.04	0.18	
1913	0.00	0.18	
1914	0.03	0.15	
1915	0.00	0.18	0.17
1916	0.03	0.11	0.14
1918	0.05	0.09	0.11

± Voluntary vaccination against typhoid fever was authorized in the army in 1909 and made compulsory in 1911.

++ Taken from the statistics of the original registration area.

A comparison of the typhoid fever mortality rate for the army with a similar age group in the civilian population for the years of 1900 to 1918 is given in Table No.8. The reduction of the civilian mortality rate shows the effect of the gradual development of sanitary measures during the years intervening between 1900 and 1918.

The residual typhoid fever remaining in the civilian group is about 10 per 100,000 and in the army, it is about 3 per 100,000. Were the sanitary conditions in each group of a similar nature, the reduction of the mortality rate in the army below 10 per 100,000 could be attributed to the effect of the vaccination. Such a deduction is dangerous, however, in that in peace times, the sanitary conditions found in the army are better than in the average civilian community. On the other hand, the sanitary conditions in time of war, in so far as the prevention of the transmission of the infection of typhoid fever is concerned, are not as good as they are in the typical civilian community and the records show that the typhoid fever death rate in the army was only 5 per 100,000 during 1918.

An analysis of the military situation during the years between 1911 and 1918 show that there was a troop movement to the Mexican border in 1911-12, in 1914, and again in 1916-17, after which the general troop movements of the

World War took place. The conditions were fairly stable in the years of 1913 and 1915. As shown in Table No. 8, these troop movements are reflected in the typhoid fever death rate. The results obtained indicate that, with a closed universe and under good sanitary conditions, it would be possible to eradicate typhoid fever by the use of universal vaccination.

The reduction in the incidence of typhoid fever in the army following the introduction of compulsory antityphoid vaccination has shown that this measure as a method of preventing the occurrence of the disease is of the greatest value; but the experience gained during the World War has shown that it cannot be regarded as a substitute for the observance of sanitary precautions. The emphasis laid upon vaccination during the early months of the war led many inexperienced medical officers to regard vaccination as an absolute protection against typhoid fever, while in reality such is far from being the case.

The Surgeon General's Report shows that small but severe epidemics of typhoid fever occurred that were directly due to the lack of proper sanitary precautions.[32]

The period from June 1, 1917, to June 1, 1918, witnessed a general troop movement to Europe. During this time only a few sporadic cases of typhoid fever occurred and second-

ary cases were rare.

In July 1918, a replacement unit consisting of 248 men from Camp Cody, New Mexico, arrived overseas with 98 cases of typhoid fever. The case fatality rate was 8.42 per cent. The men were undoubtedly exposed to massive doses of the infection at some time during the journey.

In August, 1918, a small detachment of troops stationed at Bazoilles, France, developed 15 cases of typhoid fever directly traceable to a cook who had remained on duty as cook for the detachment mess for five days after the onset of typhoid fever. The disease was endemic among the local civilian population at the time.

A number of small epidemics of this type occurred among the troops. Practically all of the divisions engaged in the Argonne offensive were exposed to massive infection by the drinking of contaminated water. In some, only a few initial cases occurred and prompt enforcement of sanitary measures prevented the occurrence of secondary cases. In other units these measures were, to a large extent, neglected because of "military necessity" and entire reliance placed upon the immunity conferred by the vaccination, with the result that many secondary cases occurred. As an example of these two extremes may be cited the...Division in which five cases occurred between October 1, 1918 and February 1,1919,

and the ...Division, in which 115 cases occurred during the same period of time, and while the two divisions were living under the same conditions except for the difference in the enforcement of the sanitary regulations. These cases included the paratyphoids.[24]

A small epidemic of 14 cases occurred at Nevers, France, in the winter of 1919 among vaccinated American soldiers. An epidemiological study ruled out water or contact as a source of the infection; and it was finally shown that the bacilli were transmitted in the ice cream sold to the soldiers in the French cafes in the city of Nevers. This epidemic, though small is of interest because it illustrates that massive doses of the infection can be transmitted by ice cream.

One of the best examples of the results of a widespread infection in a population consisting of both vaccinated and unvaccinated individuals is that of an epidemic which occurred in a community in the Hawaiian Islands in 1917.[31] The infection originated with a Japanese laborer who was employed on the water supply system while in the incubation period of typhoid fever. The results observed are tabulated in Table No.9.

Table No. 9.

Typhoid Epidemic in Hawaii, H.T., in 1917.

	Population	Number of cases of typhoid fever	Morbidity rate per M.	Mortality rate per M.
Vaccinated	4087	55	13.45	0.97
nvaccinated	812	45	55.41	8.62

The failure of vaccine to afford protection in some cases may, in a few instances, be due to a faulty technique in the administration of the vaccine, but it is believed that, in the vast majority of cases, it is due to the fact that the immunity conferred by the vaccine is relative and not absolute. Vaccination provides each individual with a barrier against a certain amount of the infection of typhoid fever; and when this amount of the infection is exceeded he develops the disease. The degree of the immunity developed in different persons must vary so that the amount ofthe infection which would produce the disease in one person would be unable to overcome the immunity in another.

If this view is correct, the prevention of typhoid fever by vaccination would also involve the maintenance of such sanitary conditions as would eliminate the opportunity for the individual to ingest at any one time a massive dose of the typhoid bacilli.

Under the conditions of military discipline vaccina-

tion can be employed as a preventive measure against typhoid fever, but the application of this measure to prevent the disease in the civilian population of a city would be a very difficult matter. The public sentiment against vaccination, the lack of public knowledge regarding the disease, and the fact that it cannot be shown that vaccination against typhoid fever will prevent the occurrence of the disease under any and all conditions, all tend to operate against the universal employment of this protective measure.

Disregarding the many difficulties that would surround any attempt to vaccinate the entire population of a city against typhoid fever and considering only the economic viewpoint of such a procedure, the value of universal vaccination is extremely doubtful.

In the years of 1910 to 1920 inclusive, other measures of combating the infection were successful in reducing the typhoid fever death rate in 57 cities of the United States from 19.59 per 100,000 population in 1910, to 3.52 in 1920. (Table No.10). The continuation of these measures will, in all probability, effect a still further reduction in the prevalence of the disease in these cities. Even if the entire population of the age group under 45 years in those cities could be vaccinated against typhoid fever, it is doubtful if the effect upon the incidence of the disease

would be great enough to justify the expenditure of the money and labor required to produce it, inasmuch as this money and labor could be expended in the fight against other infectious diseases with a reasonable expectation that much better results would be produced.

The sanitary standards that have resulted in the reduction of typhoid fever in these cities could not be lowered because of the fact that such standards are necessary in the control of other diseases; and also that the ingestion of a massive dose of the typhoid bacilli will produce the disease in many vaccinated persons.

Table No.10

Total Average Typhoid Fever Death in 57 Cities of the United States, for the Years of 1910 to 1920, inclusive.

Year	Total Population	Typhoid deaths	Typhoid death rate per 100,000.
1910	20,996,035	4,114	19.59
1911	21,545,014	3,391	15.74
1912	22,093,993	2,775	12.56
1913	22,642,972	2,892	12.77
1914	23,191,951	2,408	10.38
1915	23,740,930	2,068	8.71
1916	24,205,359	1,842	7.61
1917	24,740,068	1,647	6.65
1918	24,971,278	1,557	6.23
1919	25,526,186	987	3.83
1920	26,154,013	921	3.52

In the last analysis, the question as to whether vaccination against typhoid fever would or would not be accepted by any considerable proportion of the population of a city depends upon public opinion regarding such measures of protection against disease. The immunity conferred by vaccination against smallpox is absolute, and the public has been

in the process of being educated on this point for the past hundred years, and yet in 1909 one of the most progressive States in the Union in health matters (California) polled a one-third vote against compulsory vaccination of school children as a measure of protection against smallpox. If this be a fair test of the public sentiment regarding vaccination against disease, it will be many years before vaccination against typhoid fever will be accepted as a universal measure of protection.

Summary and Conclusions.

1. The reduction of the typhoid fever mortality rate in the larger cities of the United States during the past twenty years has, for the most part, been due to the purification of the public water supplies, and, to a somewhat less extent, to the control of the means of transmitting the infection, directly or indirectly, from the active cases or carriers to the non-immune individual.

2. The incidence of typhoid fever due to sources of infection other than the water supply is largely endemic and is derived from typhoid fever patients or carriers by means of an infected milk supply, by food other than milk, by flies, or by direct contact with the patient or carrier.

3. Many typhoid carriers are present in the population of any large city and the detection and control of all such carriers would be a very difficult matter at the present time. Their detection depends upon the epidemiological study of the cases of typhoid fever to determine the source of infection, the thorough bacteriological examination of the discharges from all convalescent typhoid fever patients, and keeping under observation all recovered cases who continue to eliminate the typhoid bacillus. The control of the carrier depends upon securing his coöperation, and the assistance of his family, in preventing the transmission of the infection to others and the enactment and enforcement of suitable laws for the purpose of controlling the nature of his employment and the recording of his movements.

4. The endemic typhoid fever can be and is being brought under control by the elimination of the means of transmitting the infection. These measures of control comprise the location, control and treatment of carriers and patients, and the supervision of the production and distribution of milk and other food supplies, together with the enforcement of sanitary regulations.

5.. The public sentiment against vaccination in general, and the fact that antityphoid inoculation does not con-

fer an absolute and permanent immunity against the disease, will tend to prevent universal immunization against typhoid fever. From an economic viewpoint, it is doubtful if universal vaccination would be practicable.

6. In the absence of practical measures which can be enforced for the detection and adequate control of all typhoid carriers, or in the absence of universal immunization against typhoid fever, it is highly improbable that endemic fever can be eradicated from the average American city under the conditions that exist at the present time.

7. The further reduction of the present urban typhoid fever rate depends upon increasing the knowledge of the public regarding the necessity for the control of typhoid carriers, the adequate supervision of the production and distribution of milk and other food supplies, the elimination of insanitary privies, and the correction of other sanitary defects.

Chart No.1.

Philadelphia;-

Typhoid Fever Death Rate per 100,000 for the years of 1903 to 1920,inclusive.

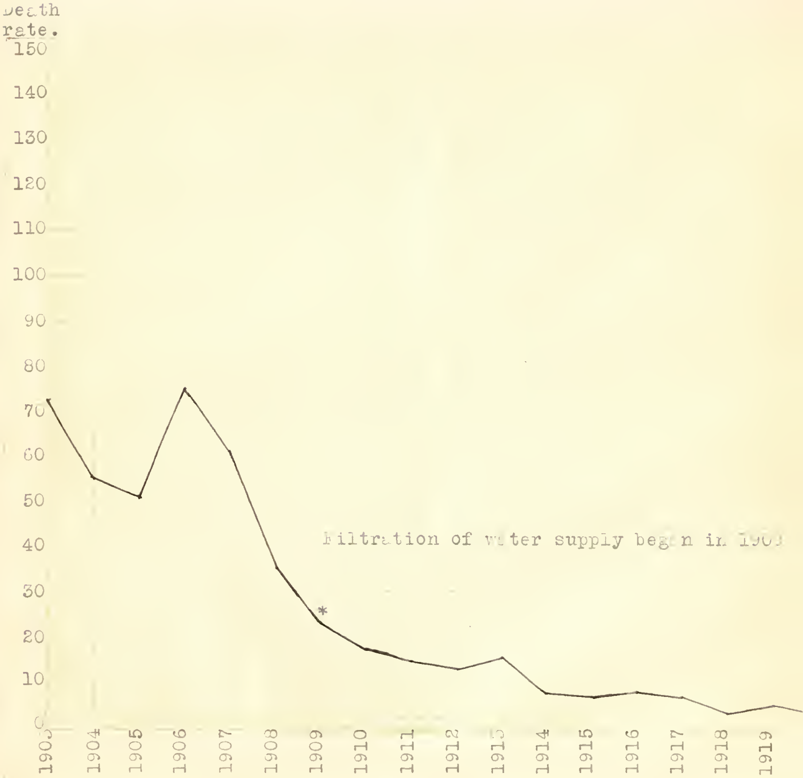
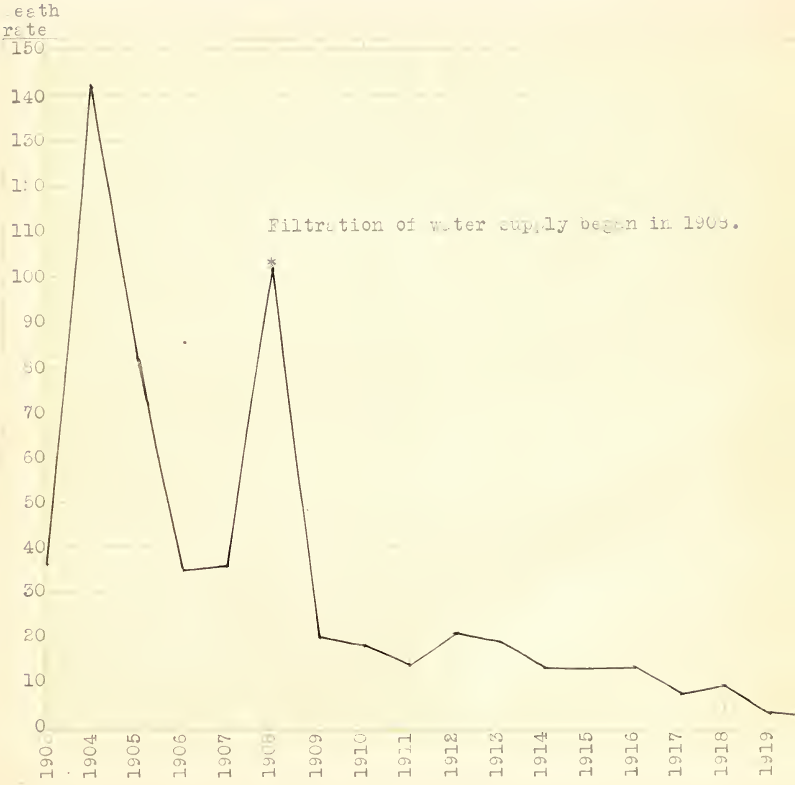


Chart No.2.

Columbus;-

Typhoid Fever Death Rate per 100,000 for the years of 1903 to 1920,inclusive.



Filtration of water supply began in 1908.

Chart No.3.

Cincinnati;-

Typhoid Fever Death Rate per 100,000 for the years of 1903 to 1920,inclusive.

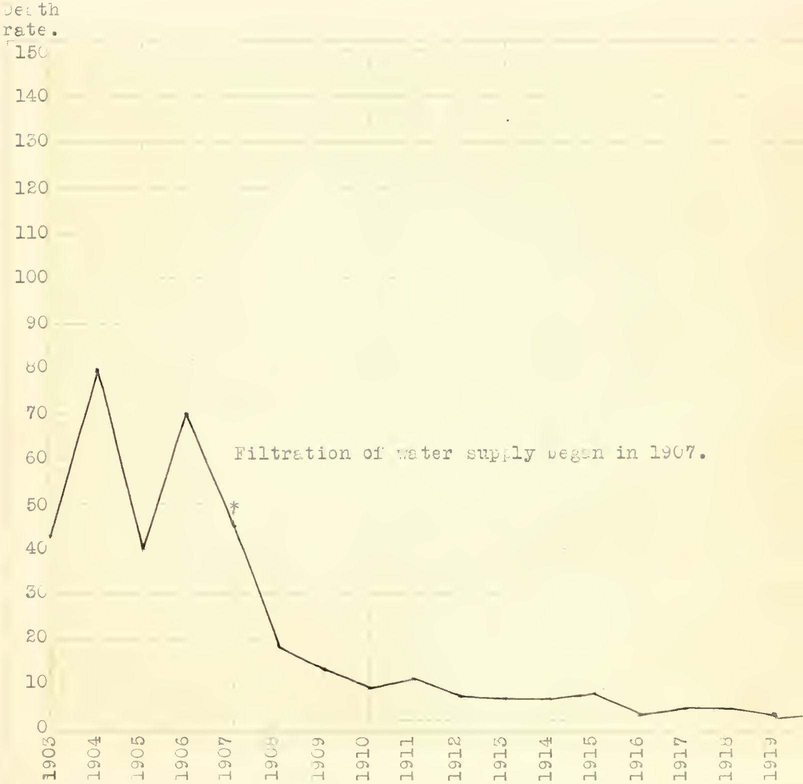
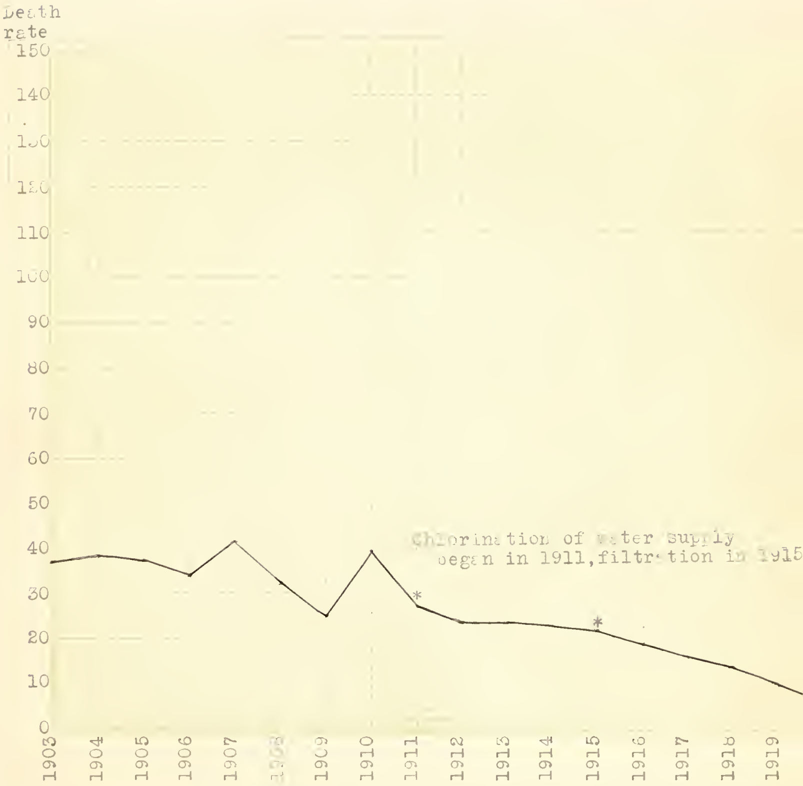


Chart No.4.

Baltimore;-

Typhoid Fever Death Rate per 100,000 for the years of 1903 to 1920,inclusive.



Chlorination of water supply began in 1911,filtration in 1915

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